Localization of Neurologic Lesions in Ruminants

Kevin E. Washburn, DVM

KEYWORDS

• Ruminant • Lesion localization • Neurologic deficits

KEY POINTS

- Localization of the origin of neurologic signs is vital to narrowing the differential list.
- Once the origin of clinical symptoms is discovered and the differential list is narrowed, further diagnostics can be more readily determined to arrive at a likely diagnosis.
- Determining the most likely diagnosis is critical because it guides prognosis and treatment regimens.
- Sometimes determining prognosis, herd ramifications, and zoonotic potential is economically and prudently more important than successful treatment and outcome.
- Although not always possible, give the animal 1 disease or condition that accounts for all clinical signs.

INTRODUCTION

The examination of a ruminant with neurologic dysfunction often presents many challenges to the diagnostician.[1–3] Challenges include the size of the animal, unruliness, economic constraints, an alarmed owner, and limited diagnostic tools that can practically be applied to the case. Therefore, the first step in these cases is to determine the most likely source of the neurologic symptoms so that an accurate prognosis and treatment regimen can be developed. There are myriad possible differentials for neurologic symptoms in ruminants that include truly primary neurologic disease, such as enzootic lymphosarcoma in the spinal canal, and secondary neurologic disease, such as hypomagnesemia. A thorough history and physical and neurologic examination are paramount to localization of the origin of neurologic signs. Once determined, the clinician can often use all the pieces of information gathered to arrive at a working diagnosis or narrowed differential list. Subsequently, it may then be possible to use only a few practical diagnostic tests, such as a cerebrospinal fluid

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Large Animal Clinical Sciences, College of Veterinary Medicine, Texas A&M University, TAMU MS 4475, College Station, TX 77843, USA
E-mail address: kwashburn@cvm.tamu.edu

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analysis, to achieve a definitive diagnosis. This article categorizes and describes common clinical signs in ruminants localizable to various regions of the central nervous system to equip clinicians with the ability to more readily determine the origin(s) of neurologic dysfunction and proceed with more discriminating diagnostic tests if warranted. Furthermore, it is hoped that this categorization can be used as an aid in answering 2 fundamental questions: Is it rostral or caudal to the foramen magnum? and Is it primary or secondary neurologic disease?

CEREBRUM

Common clinical signs localizable to the cerebrum
Opisthotonus
Blindness (with an intact pupillary light reflex)
Abnormal mentation
Change in behavior
Aimless wandering or compulsive circling
Seizures
Abnormal vocalization

Opisthotonus

Opisthotonus is defined as dorsiflexion of the head and neck. If the animal is able to sit sternal, this is sometimes referred to as *stargazing*. In the author's experience, however, ruminants with opisthotonus more frequently lie in lateral recumbency and are unable to right themselves. Ruminants with advanced tetanus or hypomagnesemia may appear to have opisthotonus as well; therefore, it is important to assess the complete neurologic and physical examination findings to determine its origin.

Blindness

Vision can, in part, be assessed from a distance as the animal is asked to navigate unfamiliar surroundings. It is important, especially with small ruminants, to assess the animals as individuals because their strong herd instincts allow them to use other heightened senses and their herd mates to navigate their environment. A complete ocular neurologic examination should follow and aid the clinician in determining whether the lack of vision is of cerebral cortical origin. Lack of vision with an intact pupillary light reflex is a hallmark of cerebral cortical disease. If the pupillary light reflex is absent unilaterally or bilaterally, the clinician should consider dysfunction of the ocular pathways or retina (vitamin A deficiency).

Abnormal Mentation

Although abnormal mentation in a ruminant can range from stupor and depression to excitement and mania, the primary determinant is how the animal is responding to its environment. Although owners may consider this to be "behavior," the natural temperament of the animal can mimic abnormalities in mentation in some cases. It is straightforward to recognize the extreme in abnormal mentation; however, the author argues that often the alteration from the normal temperament of the animal is subtle. Consequently, abnormal mentation assessment should be coupled, if possible, with the caveat of whether a change has occurred.